THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM DXM

Using the Recovery Kernel Framework

DXM · Dextromethorphan · Robo · Robotripping · Triple C · Skittles · DM · Gel Caps · Delsym

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

DXM + SSRI OR MAOI = POTENTIALLY FATAL SEROTONIN SYNDROME. CHECK YOUR MEDICATIONS.

COMBINATION PRODUCTS (DAYQUIL, NYQUIL, TRIPLE C) CONTAIN ACETAMINOPHEN OR ANTIHISTAMINES THAT CAUSE LIVER FAILURE OR CARDIAC TOXICITY AT MISUSE DOSES.

CYP2D6 poor metabolizers experience DXM completely differently — and face dramatically higher serotonin syndrome risk.

A Note Before You Begin

The Recovery Kernel Compendium addresses DXM through three separate framings that, taken together, explain both why this substance is dangerous and why it is systematically ignored by clinical systems.

First, the OTC safety illusion: 'over-the-counter doesn't mean safe for everyone.' The compendium documents this in the context of CYP2D6 pharmacogenomics, where drugs metabolized by this enzyme produce radically different effects in poor metabolizers versus normal metabolizers. DXM is the most dramatic example in common OTC use: a CYP2D6 poor metabolizer taking DXM at recommended cough suppressant doses can produce serotonin syndrome. At misuse doses, the pharmacological experience is entirely different from what a normal metabolizer experiences from the same product.

Second, the acetaminophen-glutathione depletion warning: the compendium explicitly names acetaminophen as one of the primary depletes of glutathione — the liver's master antioxidant. Many DXM-containing products also contain acetaminophen. At misuse doses, the acetaminophen load becomes directly

hepatotoxic. The person seeking DXM's dissociative effects from NyQuil or DayQuil is simultaneously delivering their liver a potentially lethal dose of a hepatotoxin.

Third, the dissociative and NMDA context: DXM's primary dissociative mechanism operates through NMDA receptor antagonism — the same system that makes ketamine therapeutically interesting and recreationally compelling. The compendium's ketamine framework, the default mode network suppression, the ego dissolution architecture, and the integration challenge all apply here — but through an OTC product that costs $8 at any pharmacy, requires no prescription, and is completely invisible to the healthcare system as a substance of misuse.

DXM misuse is the most clinically invisible significant substance problem in this guide series. The people who use it are often teenagers and young adults buying cough syrup at 2 AM. The substance is dismissed as juvenile and unworthy of serious clinical attention. The people using it are often the most isolated, most depressed, and most disconnected from any recovery support structure. This guide takes them seriously.

Read Section 0 before anything else — the product-specific toxicity hazards of DXM combinations are immediate safety information.

Section 0 — Immediate Safety: The Product Hazards

⚠ THE MOST DANGEROUS THING ABOUT DXM IS NOT DXM ITSELF — IT IS WHAT ELSE IS IN THE BOTTLE.

0.1 — Product Toxicity: What Else Is in the Bottle

THE CENTURION — The Safety Illusion of OTC Status — The Compendium's Warning Applied Directly

DXM is rarely sold alone. Most consumer cough and cold products containing DXM also contain one or more additional active ingredients that become toxic at the doses required for DXM's dissociative effects. The following table is the most important safety information in this guide — it determines whether what the person is using is primarily dangerous from DXM or from something else entirely.

Co-ingredient	Found in	Danger at misuse doses	Response if toxicity suspected
Acetaminophen (APAP / Tylenol)	NyQuil, DayQuil, many combination syrups	Hepatotoxicity (liver failure) at doses above 4g/day in adults or lower in liver-vulnerable populations. Silent initially — symptoms appear 24–72 hours after exposure.	If high acetaminophen exposure: NAC 600mg immediately. Go to emergency department if dose was very high. Liver enzyme testing. Do not wait for symptoms — liver damage is reversible only if treated early.
Chlorpheniramine (antihistamine)	Coricidin Cough & Cold (Triple C) — THE MOST DANGEROUS DXM PRODUCT	Anticholinergic toxicity at misuse doses: confusion, hallucinations, rapid heart rate, urinary retention, hyperthermia, seizure, cardiac arrhythmia. Multiple deaths specifically attributed to chlorpheniramine toxicity from Triple C misuse.	Call 911 for any suspected chlorpheniramine overdose. Anticholinergic toxicity requires hospital management — physostigmine in severe cases. This is NOT manageable at home.
Guaifenesin (expectorant)	Robitussin DM, many syrups	Severe nausea and vomiting at misuse doses. Not directly life-threatening but	Recovery position if vomiting while sedated. Hydration. Not a medical emergency in most cases

		makes high-dose DXM use extremely unpleasant and can complicate aspiration risk if vomiting while sedated.	but limits the dose that can be maintained without extreme GI distress.
Pseudoephedrine or phenylephrine (decongestant)	Sudafed PE DM, some combination cold products	Sympathomimetic toxicity: elevated blood pressure, rapid heart rate, cardiac arrhythmia, hypertensive crisis. The combination of DXM's serotonergic effects and sympathomimetic decongestants amplifies cardiovascular risk.	Chest pain, very rapid heart rate, or severe headache with decongestant + DXM: call 911. Cardiac emergency management.
DXM ONLY — the safer products	Delsym (DXM polistirex extended-release), plain DXM HBr gel capsules, Robitussin 12-Hour	DXM's own toxicity: serotonin syndrome risk (especially with SSRIs), high-dose dissociative crisis, persistent perceptual disturbances. No co-ingredient hepatic or cardiac toxicity.	From a harm reduction standpoint, if DXM is being used: pure DXM products are safer than combination products. But this is harm reduction, not endorsement.

⚠ CORICIDIN COUGH & COLD (TRIPLE C) IS THE MOST DANGEROUS COMMONLY MISUSED DXM PRODUCT. THE CHLORPHENIRAMINE CONTENT IS THE PRIMARY KILL MECHANISM, NOT THE DXM. NEVER USE TRIPLE C FOR ITS DXM CONTENT.

0.2 — Serotonin Syndrome: The Critical Drug Interaction

⚠ DXM INHIBITS SEROTONIN REUPTAKE. COMBINED WITH ANY SEROTONERGIC MEDICATION, THIS CAN PRODUCE FATAL SEROTONIN SYNDROME. THIS IS THE MOST COMMON SERIOUS MEDICAL COMPLICATION OF DXM MISUSE AND IS ENTIRELY PREVENTABLE.

DXM is a serotonin reuptake inhibitor — it blocks SERT, the same transporter that SSRIs block. Combined with any medication that also increases serotonin activity, this creates dangerous serotonin excess:

- SSRIs (Prozac/fluoxetine, Zoloft/sertraline, Lexapro/escitalopram, Celexa/citalopram, Paxil/paroxetine): all block SERT — combined with DXM = serotonin syndrome risk. Paroxetine specifically is also a potent CYP2D6 inhibitor, which means it also blocks DXM's conversion to dextrorphan, further increasing DXM plasma levels AND the serotonergic risk.
- SNRIs (Effexor/venlafaxine, Cymbalta/duloxetine): serotonin AND norepinephrine reuptake inhibition — combined with DXM = elevated serotonin syndrome risk.
- MAOIs (phenelzine, tranylcypromine, selegiline, high-dose St. John's Wort, Ayahuasca/harmaline): block serotonin breakdown — combined with DXM = severe, potentially fatal serotonin syndrome. This combination has killed people.
- Tramadol: opioid with serotonin reuptake inhibition — combined with DXM = elevated serotonin syndrome risk.
- Linezolid (antibiotic with MAOI properties): less common but documented interaction.

Serotonin syndrome recognition: Agitation, confusion, rapid heart rate, high blood pressure, dilated pupils, muscle twitching or rigidity (especially legs), diarrhea, hyperthermia, diaphoresis. This is a medical emergency. Call 911.

CYP2D6 and the serotonin syndrome amplifier: CYP2D6 poor metabolizers are at dramatically elevated serotonin syndrome risk because they cannot adequately convert DXM to dextrorphan — DXM itself accumulates to high levels, with its SERT-inhibiting properties dominating. If you know you are a CYP2D6 poor metabolizer: DXM + any serotonergic medication is a contraindication of extreme severity.

0.3 — High-Dose Emergency Recognition

Emergency	Signs	Response
4th plateau crisis / delirium	Extreme confusion, inability to communicate, loss of coordination, profound disorientation, not knowing where or who they are, possible agitation or combativeness. Differs from 3rd plateau in that the person cannot be redirected or grounded.	This is a medical emergency. Call 911. Remove from dangerous environments (traffic, heights, water). Keep calm and voice low. Do not restrain except to prevent immediate physical harm. Tell EMS specifically: 'DXM — dextromethorphan — high dose.'
Hyperthermia (overheating)	Very hot skin, stopped sweating despite heat, confusion, rapid heartbeat, muscle rigidity. DXM	911 immediately. Cool environment. Ice to neck, armpits, groin. Remove excess

	at high doses can cause hyperthermia, particularly when combined with physical activity or hot environments.	clothing. Hyperthermia is the most likely cause of DXM-related death at very high doses.
Respiratory depression (4th plateau or extreme doses)	Very slow or stopped breathing. DXM has weak opioid receptor activity that becomes clinically relevant at extreme doses — respiratory depression can occur.	Naloxone may partially help (it can reverse the opioid component of very high DXM doses) but is not a complete solution. 911. Rescue breathing if trained. Recovery position.
Seizure	More commonly from chlorpheniramine (Triple C) than DXM alone, but DXM at very high doses can lower seizure threshold.	911. Recovery position. Do not restrain. Time the seizure. Inform EMS of substance taken.

Section 1 — What DXM Did to Your Brain

THE CENTURION — The Cough Suppressant That Is Also a Dissociative, a Serotonin Reuptake Inhibitor, a Sigma Receptor Agonist, and a Weak Opioid

DXM is pharmacologically unusual. It acts on four distinct receptor systems simultaneously, and the relative contribution of each system depends on the dose, the specific product used, and — more than any other substance in this guide series — the individual's CYP2D6 genotype. Understanding this multi-system architecture explains both the unusual range of DXM's effects across the four plateaus and why the recovery must address multiple systems simultaneously.

1.1 — The Four Receptor Systems

Receptor system	What DXM does	Recovery relevance
NMDA glutamate receptors (primary — via dextrorphan)	DXM is converted by CYP2D6 to dextrorphan — which is a more potent NMDA receptor antagonist than DXM itself. Dextrorphan is pharmacologically similar to ketamine in its NMDA blockade, producing dissociation, DMN suppression, and the ego dissolution of the 3rd and 4th plateaus.	NMDA rebound on cessation: the same glutamate excitotoxicity seen in ketamine withdrawal, though typically milder. NAC and magnesium are the primary glutamate normalization supports. The compendium's ketamine framework applies to this dimension of DXM recovery.
Sigma-1 receptors	DXM itself (not dextrorphan) is a sigma-1 receptor agonist. Sigma-1 receptors are expressed throughout the brain and modulate mood, cognition, and the response to antidepressants. Sigma-1 agonism produces the unique DXM experience that is neither purely dissociative nor purely psychedelic.	Sigma-1 receptor agonism by DXM may explain some of DXM's antidepressant properties at low, non-dissociative doses. The mood crash following DXM cessation may partly reflect the loss of sigma-1 stimulation. CYP2D6 poor metabolizers, who accumulate more DXM than dextrorphan, experience sigma-1 effects more prominently.
Serotonin transporter (SERT	Both DXM and dextrorphan inhibit serotonin reuptake — a	Serotonin system recovery parallels MDMA recovery in this dimension — 5-HTP support for serotonin reconstruction, but

inhibition)	weaker mechanism than SSRIs at typical doses, but relevant at misuse doses and critical in drug interactions. The SERT component contributes to DXM's empathogenic effects at lower plateaus and to serotonin syndrome risk in combination with serotonergic medications.	with the SERT caveat that starting 5-HTP too soon after cessation (while SERT inhibition is still significant) carries serotonin syndrome risk. Wait 3–5 days after last DXM use before starting 5-HTP.
Mu-opioid receptors (weak agonism)	DXM has weak mu-opioid receptor agonism — clinically insignificant at typical doses but relevant at very high (4th plateau) doses, contributing to respiratory depression risk and to the partial naloxone response in high-dose overdose.	At therapeutic doses, the opioid component is minimal. At 4th plateau doses (1000mg+), the opioid component contributes meaningfully to respiratory depression risk. DLPA (enkephalinase inhibitor) supports the endogenous opioid system for recovery but is not a primary target in DXM recovery as it is for opioid-primary substances.

1.2 — The CYP2D6 Split: Two Different Substances in the Same Bottle

THE CENTURION — The Most Genetically Variable OTC Substance in Clinical Pharmacology

The compendium's CYP2D6 documentation states plainly: 'If you've ever felt that pain medications either didn't work or worked far more intensely than they should, CYP2D6 testing matters.' For DXM, this statement is more literal than for any other substance in the guide series. CYP2D6 doesn't just affect the intensity of DXM — it determines which pharmacological entity is primarily experienced.

CYP2D6 phenotype	What happens to DXM	Clinical and recovery implications
Normal metabolizer (~75% of Caucasians, higher in Asian populations)	DXM is efficiently converted to dextrorphan. At misuse doses, dextrorphan (the potent NMDA antagonist) is the primary active compound. The experience is predominantly dissociative — ketamine-like NMDA blockade with DMN suppression and ego dissolution. Sigma-1 and SERT components are present but the	Recovery addresses primarily the NMDA dimension (ketamine-adjacent recovery framework) with secondary serotonin reconstruction. The dissociative quality of the experience is the primary recovery target.

	NMDA dominates.	
CYP2D6 poor metabolizer (~7–10% Caucasians; ~1–2% Asians)	DXM is NOT efficiently converted to dextrorphan. DXM itself accumulates. DXM has much stronger SERT inhibition and sigma-1 agonism relative to its NMDA blockade than dextrorphan does. The experience is less purely dissociative and more empathogenic/sigma-1 at the same dose — and dramatically more serotonergic.	CRITICAL: Poor metabolizers have dramatically higher serotonin syndrome risk from DXM + any SSRI. The accumulation of DXM's SERT-inhibiting effects without adequate conversion means serotonin syndrome can occur at what a normal metabolizer would consider low doses. Poor metabolizers should NEVER combine DXM with any serotonergic medication. Test strips and cautious approach.
CYP2D6 ultra-rapid metabolizer (~1–2%)	DXM is converted to dextrorphan very rapidly — peak dextrorphan levels reached faster, potentially producing more intense and faster-onset dissociative effects at the same dose.	Less clinically documented for DXM specifically. The faster conversion means the dissociative NMDA effects may be more prominent and shorter-acting. Also: common CYP2D6 inhibiting drugs (paroxetine, fluoxetine, bupropion) convert ultra-rapid metabolizers to effectively poor metabolizers, dramatically changing the DXM experience and risk profile.
CYP2D6 inhibited by other medications	Fluoxetine (Prozac) and paroxetine (Paxil) are both SSRIs AND potent CYP2D6 inhibitors. Taking DXM while on these medications converts a normal CYP2D6 metabolizer to effectively a poor metabolizer — DXM accumulates, serotonin syndrome risk increases dramatically, AND the SSRI is adding its own SERT blockade.	DXM + Prozac or DXM + Paxil = one of the highest-probability serotonin syndrome drug combinations in OTC use. This combination has produced serious adverse events. Know your medications.

1.3 — The Four Plateaus: The Dose-Effect Architecture

THE CENTURION — The Framework That the DXM Community Built From Experience

The 'plateau' framework for DXM effects was not developed by pharmaceutical researchers but by the online DXM harm reduction community — primarily through the work of William White's DXM FAQ and subsequent community documentation. It remains the most practical dosing framework for understanding DXM's effect profile. The compendium's approach to precision language applies:

knowing which plateau the person was using determines which systems are most implicated in recovery.

Plateau	Approximate dose	Primary experience	Primary pharmacological mechanism
First plateau	100–200mg	Mild stimulation, euphoria, increased sociability, mild music enhancement. Subjectively similar to mild alcohol or cannabis intoxication for many users.	Primarily sigma-1 agonism and mild SERT inhibition. NMDA blockade minimal at this dose. This is the dose range where some antidepressant researchers have investigated DXM.
Second plateau	200–400mg	Significant intoxication, impaired coordination, altered time perception, music significantly enhanced, beginnings of dissociative effects. Strong mood elevation.	Meaningful dextrorphan accumulation (in normal CYP2D6 metabolizers) producing NMDA blockade. Serotonin system significantly affected. This is the range of compulsive recreational use for most users.
Third plateau	400–700mg	Strong dissociation, out-of-body experience, profound alterations in self-perception, visual phenomena, the 'M-hole' or 'K-hole' adjacent state. Significant coordination impairment — falls are a real hazard.	Substantial NMDA receptor blockade producing DMN suppression. Sigma-1 agonism prominent. The experience has psychedelic and dissociative qualities simultaneously — the pharmacological signature of DXM's multi-receptor profile.
Fourth plateau	700–1500mg+	Near-complete disconnection from ordinary reality. Delirium-like states. Profound ego dissolution. Communication may be severely impaired or impossible. Deep, trance-like states. Very high risk.	Very high NMDA blockade; meaningful weak opioid receptor activation begins to contribute; hyperthermia risk increases significantly; anticholinergic-like effects may emerge. This is the emergency range.

1.4 — What DXM Was Providing: The Compendium's Diagnostic Question

THE CENTURION — The Noise Went Away. Understanding What Noise Is the Recovery Target.

DXM's multi-receptor profile means it was providing different things to different users at different doses. The recovery architecture depends on which function was primary:

What DXM provided	The underlying deficit it addressed	What recovery must address
Dissociative escape — relief from ordinary self-referential consciousness	Depression, anxiety, trauma, ADHD — any state where the noise of ordinary consciousness is unbearable. The DMN suppression of high-plateau DXM is the same mechanism as ketamine's therapeutic action: temporarily disabling the self-referential loop.	See Section 6 (natural porting) and the ketamine guide's framework for natural DMN suppression. The meditation/flow/NSDR architecture applies directly.
Emotional analgesia — relief from emotional and social pain	DXM's sigma-1 agonism and serotonergic effects at first and second plateau produce genuine mood elevation and a sense of emotional warmth. For people experiencing depression, social anxiety, or loneliness — this was real relief from real pain.	Serotonin reconstruction (5-HTP, inositol). Genuine social connection. Depression and anxiety treatment through appropriate channels. The emotional pain that DXM was buffering requires direct address.
ADHD symptom management — paradoxical focus at lower doses	At first plateau doses, some people with ADHD report that DXM produces a paradoxical focusing effect. This is likely through the combined NMDA and sigma-1 modulation that affects prefrontal cortex circuits relevant to attention. DXM as ADHD self-medication is underrecognized.	ADHD evaluation and appropriate non-stimulant treatment (atomoxetine, guanfacine, viloxazine) or stimulant treatment under addiction medicine supervision. Treating the ADHD removes a primary driver of DXM use.
Consciousness exploration — accessing non-ordinary states	DXM's unique pharmacological profile produces experiences that are genuinely difficult to categorize — neither purely dissociative (like ketamine) nor purely psychedelic (like LSD) but something in between. Some users approach DXM as a tool for consciousness exploration, particularly given its OTC accessibility.	Section 6's natural porting architecture — meditation, NSDR, holotropic breathwork, contemplative practice — provides non-chemical access to the consciousness territory DXM was exploring. The LSD guide's framework for DMN suppression and ego dissolution through earned practice applies here.
Cheap, accessible, invisible intoxication	DXM's OTC status makes it uniquely accessible for people who cannot afford, cannot safely purchase, or cannot be detected using other substances	This function requires structural response: addressing the scarcity, coercive environment, or accessibility barrier that made OTC

	— particularly teenagers, people in coercive environments (schools with drug testing), and people in economic scarcity.	intoxication the best available option. Not something supplement stacks address — social and structural support.

1.5 — The Internet Community and the Knowledge It Produced

THE CENTURION — The Most Thoroughly Documented Harm Reduction Community for Any OTC Substance

DXM misuse has one of the richest online harm reduction communities of any substance — the DXM FAQ by William White (written in the 1990s and still referenced), Erowid's DXM vault, the DXM community at Reddit and various forums. This community developed the plateau framework, documented the serotonin syndrome risk decades before most clinicians acknowledged it, and produced genuine safety information that clinical medicine has largely ignored because DXM was not considered a 'serious' drug problem.

This guide honors that knowledge base while extending it with the Recovery Kernel framework's biological and genomic precision. The harm reduction community knew about CYP2D6 before most physicians did. The recovery framework adds: what happens after you stop, how to support the systems DXM damaged, and how to build the access to the states DXM was providing through means that don't come with a co-ingredient hepatotoxin.

Section 2 — Your Genomic Blueprint

DXM has the most genetically variable pharmacological profile of any substance in this guide series. CYP2D6 is the defining variable — it determines which compound primarily reaches the brain and therefore which receptor systems dominate the experience. Recovery strategy depends entirely on knowing which phenotype applies.

The DNA Testing Panel for DXM Recovery

Gene	What it controls	Key variant	DXM recovery relevance
CYP2D6	Primary DXM metabolism — converts DXM to dextrorphan (more potent NMDA antagonist)	*4/*4 poor metabolizer (7–10% Caucasians); *1/*1 normal; ultra-rapid variants	THE defining gene for DXM — more than for any other substance in this guide series. The compendium explicitly documents CYP2D6's role in OTC drug safety. For DXM: poor metabolizers experience DXM itself (sigma-1, SERT inhibition) as the primary compound rather than dextrorphan (NMDA). Their serotonin syndrome risk is dramatically elevated because DXM accumulates without conversion. Medications that inhibit CYP2D6 (paroxetine, fluoxetine, bupropion) convert normal metabolizers to effectively poor metabolizers. Get this tested before any future use of any CYP2D6-metabolized medication.
SLC6A4 / 5-HTTLPR	Serotonin transporter expression and sensitivity	Short/short allele (reduced SERT expression)	SLC6A4 short/short carriers have reduced SERT expression — DXM's SERT inhibition produces a more pronounced serotonergic effect in these individuals. Both the desired effects (emotional warmth, mood elevation at lower plateaus) AND the serotonin syndrome risk are amplified. Short/short carriers with CYP2D6 poor metabolizer status face the most extreme serotonin syndrome risk from DXM + serotonergic medications.
HTR2A	5-HT2A serotonin receptor — determines	Various variants affecting receptor	DXM's sigma-1 agonism and high-dose NMDA blockade produce experiences that engage the 5-HT2A receptor-mediated DMN suppression

	intensity of the psychedelic-adjacent component of DXM's effects	sensitivity	system similarly to psychedelics. HTR2A high-expression variants may experience DXM's upper plateau effects as more intensely psychedelic-adjacent. Also relevant to HPPD risk — persistent perceptual disturbances after heavy DXM use follow the same pathway as LSD-associated HPPD.
COMT	Dopamine and stress chemical clearance	Val158Met (rs4680)	COMT Met/Met (Sage, slow clearance) is overrepresented in DXM use for dissociative escape from anxiety. The Sage nervous system that runs chronically hot on its own stress chemicals finds in DXM's sigma-1 and NMDA mechanisms relief that the baseline anxiety management does not provide. Recovery for Sage COMT: anxiety management is the primary recovery target alongside NMDA normalization.
MTHFR	Methylation — neurotransmitter synthesis across all systems	C677T (rs1801133), A1298C (rs1801131)	MTHFR variants reduce the efficiency of dopamine and serotonin synthesis — contributing to the depression and anxiety that drove DXM use. 5-MTHF supplementation addresses the synthesis deficit and often produces meaningful improvement in the underlying mood symptoms that DXM was managing. Particularly important for the depression-driven DXM use profile.
DRD4 / DAT1 (ADHD-associated)	Dopamine signaling — novelty seeking and attention regulation	DRD4 7-repeat (rs1800955); DAT1 10-repeat (rs28363170)	The ADHD-associated variants DRD4 7-repeat and DAT1 10-repeat are specifically relevant for DXM recovery because a significant proportion of DXM users with problematic patterns have undiagnosed or undertreated ADHD. DXM's multi-receptor effects may provide ADHD symptom relief. Identifying and treating the ADHD removes a primary driver of use.

Phenotyping Without Testing

- Did DXM produce dramatically stronger effects than peers reported at the same product dose? → Possible CYP2D6 poor metabolizer — DXM

accumulating without adequate conversion. OR: CYP2D6 inhibiting medication (SSRI) converting you to effectively a poor metabolizer.

- Did DXM produce primarily emotional warmth and mood elevation rather than dissociation? → Sigma-1 and SERT components dominant over NMDA — suggests CYP2D6 poor metabolizer profile (DXM itself more than dextrorphan).
- Did you ever experience a serotonin syndrome-like reaction with DXM? → CYP2D6 poor metabolizer + SLC6A4 short/short + likely concurrent serotonergic medication.
- Did DXM at lower doses (1st plateau) seem to help you focus or quiet ADHD-like symptoms? → DRD4 7-repeat + DAT1 10-repeat ADHD profile. ADHD evaluation is a recovery priority.
- Did you develop persistent visual disturbances or HPPD-like phenomena after heavy DXM use? → HTR2A high-expression variant. Section 4.4 addresses HPPD management from heavy DXM use.

Section 3 — The Recovery Framework: What DXM Recovery Actually Requires

THE CENTURION — Like Ketamine, Like LSD — But Available at Every Pharmacy. The Invisibility Problem.

DXM does not produce the seizure-risk physical withdrawal of alcohol and benzodiazepines. It does not produce the medical emergency of opioid withdrawal. What it produces is closer to the ketamine and LSD recovery territory — NMDA rebound, serotonin system disruption, HPPD risk, integration backlog for high-plateau experiences, and psychological dependence on the dissociative or altered states.

The specific challenge of DXM recovery that no other guide in this series faces: the substance is OTC, costs almost nothing, and can be purchased at any pharmacy without age verification in most states. Environmental redesign — removing the supply — requires genuinely different strategies than removing an illicit drug supply. The access barrier is essentially zero, which means recovery must be substantially internal rather than primarily environmental.

3.1 — What Makes DXM Recovery Different

Challenge	Why it's unique to DXM	Recovery response
OTC accessibility	A decision to use can be executed in 10 minutes with $8 at any pharmacy, gas station, or grocery store. The environmental barrier to relapse is essentially zero — every errand potentially passes a supply source. This is uniquely different from illicit drugs where supply acquisition requires specific contacts and effort.	Internal recovery architecture is primary: the supplement stack that reduces the neurochemical craving, the natural dissociative access that reduces the functional pull, and the mindfulness that recognizes the craving before it converts to a purchase. Some people ask pharmacy staff to flag their account — this is available at some chain pharmacies.
Clinical invisibility	DXM is not recognized as a primary substance of misuse in most clinical settings. Many healthcare providers are unaware of DXM misuse beyond the pediatric context. The person who discloses DXM use may be dismissed, minimized, or receive an 'it's just cough syrup' response that invalidates their experience and the real complications they are	Protocol 13 (The Physician Conversation) requires specific framing for DXM: 'I have been misusing dextromethorphan at doses that produce dissociative effects, and I am experiencing NMDA rebound symptoms, serotonin system disruption, and persistent perceptual disturbances.' The clinical specificity makes dismissal less possible.

	managing.	
Adolescent context	DXM misuse is heavily concentrated in adolescents and young adults. Recovery in this age group may involve family systems, school environments, and developmental factors that standard adult addiction recovery frameworks do not address. The person may not have had their substance use problem taken seriously by adults around them precisely because it's 'just cough syrup.'	Family involvement where appropriate and where safety allows. School counselor or adolescent therapist with DXM knowledge. The specific developmental tasks of adolescence (identity formation, peer relationships, academic performance) are recovery targets alongside the neurochemical repair.
Product toxicity variation	Not all DXM misuse involves the same product. The person who has been using pure DXM gel capsules faces different recovery from the person who has been drinking NyQuil for the DXM (acetaminophen co-exposure) or using Triple C (chlorpheniramine toxicity).	Medical assessment must be product-specific. Liver function testing (AST, ALT) for anyone with significant acetaminophen co-exposure. Cardiac assessment for anyone with decongestant co-exposure.

3.2 — The Withdrawal and Aftermath Timeline

Timeline	Biology	Experience	What to do
Hours 0–24 (immediate after)	NMDA receptors hypersensitive (as in ketamine recovery); glutamate rebound; serotonin system disruption from SERT inhibition; possible sigma-1 receptor rebound	Anxiety, cognitive hyperactivity, sensory hypersensitivity, possible visual phenomena (HPPD-like), mood crash, possible craving. Sleep disruption.	NAC and magnesium immediately — glutamate homeostasis and NMDA channel support. No stimulants. Low sensory environment. Rest. The 4-7-8 breath protocol.
Days 1–7	NMDA receptor sensitivity normalizing; serotonin system rebuilding; sigma-1 receptor density recovering; dopamine secondary recovery beginning	Depression (often significant — the mood elevation of DXM has cleared but the natural system hasn't recovered), anxiety, cognitive fog, possible persistent visual phenomena, sleep disruption,	Full supplement protocol (Section 4). 5-HTP after Day 3–5 (not immediately — allow SERT recovery before adding serotonin precursor). Exercise begins. The depression is the most common relapse driver in this window.

		irritability	
Weeks 2–8	All systems gradually recovering; HPPD if present either diminishing or being assessed; the underlying conditions (ADHD, depression, anxiety) that DXM was managing reasserting without chemical buffer	Progressive improvement with variability; depression and anxiety may be significant if underlying and untreated; HPPD symptoms if present; OTC craving triggered by any pharmacy visit or reminder	Begin underlying condition treatment (ADHD, depression, anxiety). HPPD management if persistent. The natural access architecture of Section 6 building. Community — the profound isolation that DXM use often involves requires deliberate counter-force.
Months 1–6	Substantial NMDA recovery; serotonin and sigma-1 systems rebuilt; underlying conditions being managed; HPPD resolved for most users	Meaningful recovery; genuine mood and cognitive capacity returning; the states DXM was providing beginning to become accessible through meditation, exercise, and contemplative practice; relapse risk primarily at low-barrier moments (pharmacy access, depression dip)	The long-term work: the underlying depression, ADHD, or anxiety management that is now the central recovery target. DXM was the solution to a real problem — the problem still needs solving through sustainable means.

Section 4 — Biological Reconstruction

DXM recovery requires concurrent reconstruction of three systems: the NMDA glutamate system (like ketamine recovery), the serotonin system (like MDMA recovery but milder), and the liver if acetaminophen co-exposure has occurred (urgent if significant). The methylation foundation underlies all three.

4.1 — The Methylation Foundation

Supplement	Dose	Timing	DXM-specific notes
5-MTHF (L-methylfolate)	400–1000mcg daily	Morning with food	Serotonin synthesis support — relevant to DXM's SERT component. For MTHFR variant carriers whose depression drove DXM use, 5-MTHF often produces meaningful early improvement in mood — addressing the methylation deficit that the sigma-1 and SERT mechanisms of DXM were compensating for.
Methylcobalamin (B12)	1000–2000mcg sublingual	Morning	Active B12 for methylation support. Heavy DXM use — particularly with combination products — is associated with poor nutrition during use periods. B12 repletion is foundational.
P5P (active B6)	25–50mg	Morning	Cofactor for serotonin, dopamine, and GABA synthesis. The concurrent disruption of serotonin (SERT inhibition by DXM) and glutamate (NMDA blockade) places particular demand on P5P-dependent enzymatic pathways during recovery.
Magnesium glycinate	400–600mg elemental	Morning and evening	The endogenous NMDA channel modulator. As in ketamine recovery: magnesium provides natural NMDA channel blockade that reduces the glutamate rebound without producing dissociation. The most important supplement for the NMDA dimension of DXM recovery. Begin Day 1.

4.2 — URGENT: Liver Support for Acetaminophen Co-Exposure

⚠ IF YOU HAVE BEEN USING DXM PRODUCTS THAT ALSO CONTAIN ACETAMINOPHEN (NYQUIL, DAYQUIL, COMBINATION SYRUPS): NAC IS AN URGENT PRIORITY, NOT OPTIONAL SUPPLEMENTATION. ACETAMINOPHEN TOXICITY IS SILENT FOR 24–72 HOURS AND THEN BECOMES IRREVERSIBLE.

The compendium specifically names acetaminophen as one of the primary deplete of glutathione. NAC (N-Acetyl Cysteine) provides the cysteine required to synthesize glutathione — the liver's primary defense against acetaminophen hepatotoxicity.

- NAC 600–1200mg orally immediately if significant acetaminophen-containing DXM product use occurred in the past 24 hours. This is the same mechanism as the hospital IV NAC used in acetaminophen overdose — oral supplementation works for lower-level exposure.
- If high-dose acetaminophen exposure (more than 4g in 24 hours at therapeutic doses, lower in liver-vulnerable populations): go to an emergency department. IV NAC is more effective than oral at high exposure levels. Do not wait for symptoms.
- Liver function testing (AST, ALT, bilirubin): if any significant acetaminophen co-exposure history, liver enzymes should be checked to establish the current hepatic status.
- Ongoing liver support: milk thistle (silymarin 200–400mg daily), alpha-lipoic acid (300mg daily), NAC (600mg morning and evening) for 4–8 weeks after cessation if acetaminophen exposure was significant.

4.3 — NMDA System Normalization

From the ketamine guide, applied to DXM recovery:

- NAC (600–1800mg daily, divided, with food): restores glutamate homeostasis in the nucleus accumbens. The glutamate rebound of NMDA antagonist withdrawal responds to NAC's cystine-glutamate antiporter modulation. Begin immediately.
- Magnesium glycinate (400–600mg elemental, morning and evening): natural NMDA channel blockade providing gentle modulation of the rebound glutamate surge.
- L-Theanine (200–400mg, as needed): partial NMDA modulation at the glycine site; reduces the cognitive hyperactivity and anxiety of the NMDA rebound period.

- Glycine (3–5g, evening): NMDA co-agonist supporting receptor normalization during sleep. Counterintuitively, supporting some NMDA activity helps the receptor system normalize after chronic antagonism.
- Omega-3 DHA (2g+ daily): neuronal membrane support for NMDA receptor environment; anti-neuroinflammatory.

4.4 — Serotonin Reconstruction

⚠ START 5-HTP ONLY AFTER 3–5 DAYS OF DXM CESSATION. EARLIER ADDITION RISKS SEROTONIN SYNDROME FROM RESIDUAL SERT INHIBITION BY DXM COMBINED WITH ADDED SEROTONIN PRECURSOR. THE KETAMINE GUIDE'S CAUTION APPLIES HERE.

- 5-HTP (50–100mg, evening — after Day 3–5): serotonin precursor for the SERT-inhibition dimension of DXM's disruption. Do not combine with any SSRI, SNRI, or MAOI.
- L-Tryptophan (500–1000mg, evening, away from protein): alternative to 5-HTP — upstream serotonin precursor, gentler onset. Use one OR the other.
- Inositol (4–8g, divided): serotonin receptor sensitivity modulation; anti-anxiety; relevant to the social anxiety that often underlies DXM use.
- EPA-dominant omega-3 (2–4g daily): anti-inflammatory and mood support for the serotonin recovery dimension.

4.5 — HPPD Management from Heavy DXM Use

Heavy or long-term DXM use at 3rd and 4th plateau doses can produce HPPD-like visual phenomena — the same category of perceptual disturbances documented with ketamine and psychedelic use. The compendium names HPPD in the context of hallucinogen use; DXM's NMDA and sigma-1 mechanisms place it in this category for HPPD risk.

- Complete cessation of DXM: NMDA and sigma-1 receptors cannot recover density and sensitivity under continued stimulation. This is the prerequisite.
- Cannabis cessation: the same HPPD exacerbation documented with psychedelic-associated HPPD applies to DXM-associated visual phenomena. Cannabis is the most reliably reported worsening factor.
- The HPPD management protocol from Section 4 of the LSD guide applies: the antioxidant neuroprotective stack (EGCG, alpha-lipoic acid, NAC, vitamin C, DHA), sleep optimization, anxiety management, and avoidance of SSRI monotherapy (which may worsen visual phenomena).
- If visual phenomena are persistent and impairing at 4+ weeks after cessation with cannabis also ceased: neurological and psychiatric evaluation. Clonazepam (short-term) and lamotrigine have evidence for HPPD management.

4.6 — Underlying Condition Treatment

THE CENTURION — DXM Was a Solution. Recovery Requires Finding the Actual Problem.

The compendium's C-37 (Substance Masking Analysis) is directly applicable here: 'every substance preference is a signal, and every signal points to something that needs repair.' For DXM use disorder, the signal most commonly points to:

- Depression: often the primary driver, particularly in adolescent and young adult populations. 5-MTHF evaluation first (MTHFR-variant depression is extremely common and responds to 5-MTHF without requiring SSRI prescription). If clinical depression persists at 6 weeks post-cessation with full supplement protocol: psychiatric evaluation. SSRIs are appropriate but require full cessation of DXM — never combine.
- ADHD: undiagnosed ADHD is significantly overrepresented in DXM use disorder. ADHD evaluation should occur early in recovery. Non-stimulant treatment (atomoxetine, guanfacine, viloxazine) is appropriate for people with stimulant use concerns alongside DXM. Stimulant ADHD treatment requires careful risk-benefit assessment with addiction medicine involvement.
- Social anxiety disorder: DXM's sigma-1 and serotonergic effects provide social ease and anxiety dissolution that is specifically compelling for people with severe social anxiety. Buspirone (non-addictive), CBT, and SSRI-based treatment (after complete DXM cessation) address the underlying condition.
- Trauma and PTSD: DXM's dissociative effects provide trauma buffering — the same mechanism as ketamine's therapeutic dissociation from traumatic memory. EMDR, CPT, or other trauma-specific treatments alongside the recovery framework.

4.7 — Sleep Reconstruction

- Magnesium glycinate (400mg elemental, evening): NMDA modulation and GABA support.
- Glycine (3–5g, 30 min before bed): core body temperature reduction and NMDA receptor support.
- L-Theanine (200mg, evening): alpha-wave promotion and NMDA modulation.
- Melatonin (0.5–1mg sublingual, consistent time): circadian signal for disrupted sleep architecture.
- 5-HTP (50mg, evening, after Day 3–5): serotonin-melatonin conversion supporting sleep.

The compendium's psychedelic/dissociative sonic protocol for sleep: ambient, drone, theta binaural beats (4–8 Hz); very low volume; no sudden dynamics. The

DXM-specific note: avoid music from the using context — the auditory memory is potent and can trigger craving.

4.8 — Peptide Support

Peptide	Why it matters for DXM recovery	Timing	Notes
BPC-157	NMDA receptor modulating properties; dopamine and serotonin normalization; gut-brain axis repair; liver tissue repair (relevant for acetaminophen co-exposure cases)	Day 1 onwards — daily	BPC-157's multi-system coverage addresses the three primary DXM recovery targets (NMDA, serotonin, and liver) simultaneously. Its documented liver tissue repair properties are specifically relevant for acetaminophen co-exposure cases. Oral form is appropriate and convenient.
Selank	Anxiolytic via GABA and serotonin modulation; non-addictive; specifically addresses the anxiety that both (a) drove DXM use and (b) the NMDA rebound anxiety of cessation	Weeks 1–8 as needed	For anxiety-driven DXM use (the Sage COMT profile that found in DXM's sigma-1 and NMDA effects relief from chronic anxiety), Selank provides non-addictive serotonergic anxiolytic support during recovery. Intranasal.
Semax	BDNF upregulation; cognitive enhancement; dopamine modulation — DXM's NMDA blockade and the ADHD profile both produce prefrontal cortex impairment that BDNF-driven repair addresses	Week 2 onwards	The cognitive fog of DXM recovery — impaired attention, word-finding difficulty, slowed processing — reflects prefrontal cortex function impairment from NMDA blockade and underlying ADHD if present. Semax's BDNF-stimulating effects support both simultaneously. Intranasal.

Section 5 — The DXM Craving — Understanding and Interrupting

THE CENTURION — The Craving That Can Be Executed in 10 Minutes at Any Pharmacy — the Architecture of the OTC Problem

DXM cravings have a specific structure produced by the OTC accessibility: the decision-to-use-to-having-the-substance pipeline is radically shorter than any other substance in this guide series. An opioid craving requires finding a supply. A stimulant craving requires a dealer or diversion source. A DXM craving requires being near a pharmacy and having $8. The craving interrupt must work in the 10-minute window between trigger and pharmacy entry.

DXM Craving Trigger Map

Trigger	The underlying state	The interrupt
Passing a pharmacy or store with a cough & cold aisle	The most common and most OTC-specific trigger. The physical proximity to the supply fires the craving circuit before any conscious decision is made.	Alternative routes that avoid pharmacies during early recovery. Where unavoidable: commit to not entering the cough & cold aisle before entering the store. If possible, have a trusted person accompany during pharmacy visits in early recovery. The 10-minute rule: if a craving fires, wait 10 minutes before any purchase decision.
Depression dip	The depression that drove DXM use is the most reliable craving trigger. When mood drops below threshold — particularly in the NMDA rebound and serotonin-deficit early recovery period — the brain's trained solution is the substance that reliably elevated mood.	The depression protocol must be active and real: 5-MTHF, exercise, light exposure, social contact, possibly psychiatric treatment. The craving is pointing at the depression. The supplement and behavioral protocol addresses the depression directly, which reduces the craving frequency at its source.
Isolation — being alone with no stimulation	DXM use was often solitary. The state of being alone with no engaging activity is a strong conditioned cue — the brain learned that this state precedes DXM use. Boredom and loneliness are primary DXM triggers.	Schedule is the primary environmental intervention. The compendium's Protocol 05 for DXM recovery focuses on filling the unstructured time that DXM use occupied. ADHD management — if untreated ADHD is creating the

		intolerable boredom — is the structural solution.
Anxiety spike — particularly social anxiety	DXM's sigma-1 agonism provided social ease and anxiety relief. The return of anxiety in social situations — particularly for people with underlying social anxiety disorder — fires the DXM craving as the remembered solution.	4-7-8 breathing immediately. Selank if using peptides. Social anxiety treatment (buspirone, CBT, SSRI after complete DXM cessation). The anxiety is the signal — address the anxiety, not just the craving.
Online community / triggers	DXM has an active online community with detailed trip reports and experience documentation. Engagement with this content — even out of curiosity — can activate the craving through the visual and narrative cues.	Protocol 05 includes digital environment: specific DXM-associated communities, forums, and content. Consider temporary blocks on these during early recovery. The curiosity is normal; the engagement with the content activates the craving circuit.

THE DXM CRAVING INTERRUPT — 5-MINUTE PROTOCOL (WITHIN THE 10-MINUTE WINDOW)

1. PAUSE THE ROUTE. If walking toward a pharmacy or store: stop physically. Pause. Do not continue walking toward the supply. The 10-minute rule: the decision to purchase must not happen for 10 minutes after the craving fires. This is the window the protocol operates in.
2. EXECUTE 4-7-8 BREATHING — FIVE CYCLES. The OTC craving fires fast because the supply is near. The vagal brake is the fastest available counter-signal. Five cycles while standing still or redirecting.
3. TAKE NAC AND MAGNESIUM (or note them for next dose). The NMDA and glutamate substrate of the craving is addressed by these supplements. The craving has a biochemical component; address it biochemically.
4. NAME THE UNDERLYING STATE. Depression? Anxiety? Boredom/isolation? ADHD? The specific underlying state is the signal the craving is carrying. Naming it activates the specific intervention rather than the generic craving response.
5. CHANGE YOUR PHYSICAL ROUTE OR ENVIRONMENT. If the trigger was pharmacy proximity: take a different route. Do not enter the building. Physical environment change is the most direct intervention for the OTC-proximity trigger.
6. CONTACT. Text or call your support structure. Name what triggered it. The OTC craving in isolation is particularly dangerous because the entire use cycle — trigger, purchase, use — can happen alone and invisibly. Contact breaks the isolation.

7. EXECUTE YOUR NATURAL DISSOCIATIVE/MOOD ACCESS. From Section 6: NSDR, meditation, vigorous exercise, music absorption. The craving is for a state — provide an alternative path to an adjacent state.

Section 6 — Porting the Addiction: Building Natural Access to the States DXM Provided

THE CENTURION — Centurian Paper C-28 Applied to DXM — The Silence That Was Available All Along

DXM's multi-receptor profile provided different things for different users. The recovery architecture's natural porting matches the specific function:

- For the dissociative escape seeker: the ketamine guide's natural porting framework — meditation for DMN suppression, NSDR/yoga nidra for hypnagogic dissociation-adjacent states, holotropic breathwork for non-chemical altered states.
- For the emotional analgesic seeker: the MDMA guide's natural porting framework — genuine human connection for oxytocin and serotonin activation, exercise for endorphin and serotonin co-release, community.
- For the consciousness explorer: the LSD guide's contemplative tradition framework — any systematic path with a map of non-ordinary states and a community for navigating them.
- For the ADHD symptom manager: ADHD treatment — non-stimulant medication, behavioral strategies, structured environment, the compendium's dopamine architecture. Finding the activity that produces genuine, sustainable focus: music, martial arts, coding, any craft at the challenge-skill edge.

The Natural Architecture for the States DXM Provided

What DXM provided	Natural alternative	How to implement	For DXM recovery specifically
Dissociative escape — DMN suppression, noise quieting	Meditation (absorption states); NSDR; holotropic breathwork	Daily practice — minimum 20 minutes. Dharma Recovery (Protocol 04). NSDR: 20–30 min daily, lying still, guided attention. Holotropic breathwork with guide.	DXM's NMDA mechanism produced the same DMN suppression that ketamine does and that meditation achieves through trained attentional practice. The DXM user has been to this territory. The practice builds the path back that doesn't cost $8 and liver tissue.
Mood elevation and emotional warmth (sigma-1 + SERT)	Exercise; genuine social connection; laughter; music	Daily vigorous exercise. Scheduled social time with safe people. Comedy and play. Active music listening at high	The sigma-1 and serotonin components of DXM's mood effects are accessible through the natural serotonin and endorphin architecture. These take longer to develop than DXM

		quality.	but build rather than deplete.
Social anxiety dissolution	CBT for social anxiety; buspirone; practiced exposure; community	CBT-based social anxiety program (MBSR, CBT groups, individual therapy). Graduated social exposure — starting with smaller, safer contexts. Buspirone (prescription, non-addictive).	The social ease that DXM's sigma-1 and serotonergic effects provided is accessible through the nervous system's own pathways with practice. The practice is deliberate social exposure in progressively challenging contexts. It is slower than a pill and permanently yours.
Consciousness exploration	Contemplative tradition; float tanks; breathwork; meditation depth	One contemplative tradition practiced systematically. Monthly float tank sessions. Holotropic breathwork periodic. Daily meditation building toward absorption states.	DXM's multi-receptor state produces experiences that bridge dissociative and psychedelic phenomenology. Contemplative traditions have been mapping this territory for millennia. The practices provide sustainable access to states that are recognizably related to what DXM produced — without NMDA rebound or liver concerns.
ADHD symptom management — focus and noise reduction	ADHD treatment (medication + behavioral); meditation; structured environment; exercise	Formal ADHD evaluation and appropriate non-stimulant or stimulant treatment. Daily vigorous exercise (30–45 min) which has the strongest non-pharmacological evidence for ADHD symptom management. Structured schedule and environment.	If DXM was primarily managing ADHD symptoms, treating the ADHD removes the primary driver of use. This is both the simplest and most commonly missed intervention in DXM recovery. ADHD treatment plus the recovered natural dopamine system produces the focus DXM was providing through a healthier mechanism.

The Adolescent-Specific Recovery Architecture

THE CENTURION — The Person Who Found DXM at 15 Is Different From the Person Who Found Heroin at 30

DXM is heavily an adolescent and young adult substance. The recovery architecture for this age group has specific features that standard adult addiction recovery frameworks do not address:

- Identity formation: adolescent recovery occurs alongside the fundamental developmental task of identity formation. The person is not recovering a pre-substance identity — they may not have had one fully formed before DXM

entered the picture. Recovery is partly identity construction for the first time.

- Family system: for adolescents and young adults living with family, the family system is part of the recovery environment. Family members who understand DXM's pharmacology — and who understand that it is not 'just cough syrup' — can provide meaningful support. Family therapy is often appropriate.
- School and academic context: the cognitive fog of DXM recovery, particularly if ADHD is unmanaged, directly affects academic performance. Academic accommodation, ADHD support services, and teacher/counselor collaboration are recovery tools.
- Peer environment: the peer relationships that formed around DXM use require the same renegotiation as any substance-use social environment. Finding peers who don't use — through sport, arts, academic teams, hobby communities — is the primary social recovery intervention for adolescents.
- Developmental acceleration: some adolescent DXM users have used the substance's dissociative and consciousness-altering properties to navigate developmental experiences (sexuality, identity, family stress, existential questions) that they did not have adult frameworks to manage. Recovery includes building these frameworks — through therapy, contemplative practice, and genuine community.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	DXM-specific notes
Liver and Safety Assessment (Days 0–7)	Physician; liver function testing; Section 0 of this guide	Acetaminophen co-exposure assessment; NAC urgency; HPPD baseline; underlying condition screening	If any acetaminophen co-exposure: NAC immediately and liver function testing in the first week. This is the only DXM recovery intervention with time-urgency comparable to the medical emergencies in other guides. Everything else can be approached more gradually; hepatic assessment cannot.
Biological Foundation (Days 1–14)	Protocol 07, Protocol 09, Protocol 17	NAC (NMDA + liver), magnesium, 5-MTHF, sleep stack, L-theanine, glycine	NAC on Day 1 — it serves double duty (NMDA glutamate homeostasis AND liver support). Magnesium on Day 1. 5-MTHF on Day 1. 5-HTP: wait until Day 3–5 minimum after last DXM use to avoid serotonin syndrome from residual SERT inhibition. Sleep stack begins immediately.
Genomic Mapping (Weeks 1–4)	CYP2D6 testing; SLC6A4; COMT; MTHFR; DRD4/DAT1	CYP2D6 metabolizer status; ADHD genetic markers; serotonin system; methylation	CYP2D6 is the highest-priority genetic test in DXM recovery — it explains why the experience was what it was and informs safety for any future medication. The ADHD genetic markers (DRD4, DAT1) determine whether ADHD evaluation is a priority.
Underlying Condition Treatment (Weeks 2–12)	Psychiatry; ADHD specialist; CBT; Section 4.6	Depression treatment; ADHD diagnosis and management; social anxiety treatment; trauma work if indicated	This is the most important medium-term recovery track for DXM. The substance was solving a real problem. Identifying and treating the problem through appropriate clinical means removes the primary functional driver of use. Without this track, the recovery is holding a gap without filling it.
HPPD Management (Weeks 1–8 if present)	Section 4.5 of this guide; physician if severe	Cannabis cessation; antioxidant stack; sleep; anxiety management	DXM-associated HPPD is generally milder than psychedelic-associated HPPD but follows the same management principles. The majority of cases resolve within weeks to months of cessation. Cannabis cessation is the most important single intervention.

Natural Access Architecture (Months 1–6)	Protocol 04 (meditation), Protocol 03 (exercise), Section 6 of this guide	Daily meditation/NSDR; vigorous exercise; community; the specific state-access architecture matching the DXM function	The natural access architecture must be built to match the specific function DXM served. Dissociative escape: meditation + NSDR. Mood elevation: exercise + connection. Social ease: CBT + practiced exposure. ADHD focus: ADHD treatment + exercise. Match the architecture to the signal.
Identity and Long Game (Months 3+)	Protocol 20, Protocol 21, C-28; contemplative tradition if appropriate	Who this person is without DXM's states; the life the recovered nervous system can inhabit	For adolescent and young adult DXM users: this is partly a first construction of identity rather than a recovery of a prior one. The contemplative tradition, the creative practice, the genuine community — these are not replacements for DXM. They are the actual project that DXM was deferring.

Section 8 — The Medical Context: The Invisible Problem

DXM misuse is systematically underserved by clinical medicine for a specific reason: it is not illegal, not perceived as serious, and is associated with adolescents who are often not the primary clients of addiction treatment systems. There is no MAT for DXM. There are no DXM-specific clinical guidelines. Most physicians have minimal training in DXM misuse beyond the pediatric poison control context.

What this means for people seeking help: you may need to advocate for the seriousness of your experience. The compendium's Protocol 13 (The Physician Conversation) applies directly.

8.1 — Medical Priorities in DXM Recovery

- Liver function: AST, ALT, bilirubin, and GGT for any acetaminophen co-exposure history. Baseline and 3-month follow-up. Mild elevation is common; significant elevation requires hepatology referral.
- CYP2D6 testing: the most actionable single pharmacogenomic test for DXM history. Also informs future medication safety for any CYP2D6-metabolized prescription drugs.
- ADHD evaluation: formal psychiatric evaluation if ADHD is suspected. This is both a recovery support and a patient safety intervention — untreated ADHD drives relapse across multiple substances.
- Depression and anxiety: PHQ-9 and GAD-7 screening tools provide initial assessment. Referral to psychiatrist or psychologist for formal diagnosis and treatment planning.
- HPPD evaluation: if persistent visual phenomena are present at 4+ weeks post-cessation, neurological evaluation to rule out other causes. HPPD-experienced psychiatrist or neurologist is the appropriate referral.
- Cardiac evaluation: if decongestant-containing products were used at misuse doses — ECG and blood pressure assessment.

8.2 — Having the Physician Conversation

When seeking clinical help for DXM recovery, the framing matters. 'I've been drinking a lot of cough syrup' typically produces dismissal. The following clinical language is more likely to generate appropriate response:

'I have been misusing dextromethorphan (DXM) at doses producing dissociative effects — in the range of [X] mg per session, [frequency]. I am experiencing [specific symptoms: NMDA rebound anxiety / serotonin system disruption / persistent visual phenomena / mood disorder]. I am concerned about hepatotoxicity from acetaminophen co-exposure and want liver function testing. I would like CYP2D6 testing to understand my pharmacogenomic risk. And I believe

I have comorbid ADHD/depression/anxiety that was driving the use and needs its own treatment.'

That paragraph transforms the clinical encounter from dismissal to treatment planning.

8.3 — Purchasing Restrictions and Harm Reduction Resources

- DXM purchase restriction: many states require age verification (18+) for DXM purchases. Some chain pharmacies have implemented voluntary purchase tracking programs. CVS Pharmacy limits DXM purchases and requires ID. These restrictions are harm reduction measures — for people in recovery, they can provide an additional environmental barrier.
- DXM-specific harm reduction: Erowid's DXM vault (erowid.org/chemicals/dxm) remains the most comprehensive harm reduction information available. The DXM FAQ by William White is foundational.
- SAMHSA's Treatment Locator (findtreatment.gov): while DXM is rarely listed as a primary substance, substance use disorder treatment for comorbid depression, ADHD, and anxiety is appropriately addressed through the standard treatment locator.
- Teen Line: 1-800-852-8336 (for adolescents specifically) — peer support by teens trained in crisis response.
- Crisis Text Line: Text HOME to 741741

A Final Word

THE CENTURION — The Cough Suppressant That Told You the Truth About What You Needed

DXM was never supposed to do what it did for you. Bayer invented it to suppress coughs. It ended up in the hands of teenagers who discovered that it quieted something much louder than a cough — the noise of ordinary consciousness, the weight of untreated depression or ADHD, the unbearable social self-consciousness of anxiety, the existential questions that adolescence asks without providing any frameworks for answering them.

The compendium's C-37 principle is exactly applicable: every substance preference is a signal. The DXM preference was one of the clearest signals in this series because the signal was so specific. The NMDA blockade that quieted the mind, the sigma-1 agonism that lifted the mood, the serotonergic warmth that dissolved the social anxiety — these were not random recreational effects. They were pharmacologically precise solutions to neurobiological problems that the healthcare system had either not identified or had not adequately treated.

The OTC status that made DXM so accessible made those problems invisible too. Nobody takes an $8 cough syrup habit seriously. Nobody asks a teenager buying Robitussin what's going on at home, at school, in their head. The substance solved the problem invisibly and the problem remained invisible.

Recovery names the problem. The depression — real, biological, often MTHFR-variant impaired synthesis that 5-MTHF will address where SSRIs couldn't. The ADHD — the attention system running on the wrong frequency, for which appropriate treatment produces the focus DXM was supplying through receptor antagonism. The social anxiety — the nervous system that finds social exposure genuinely dangerous, for which CBT and practiced exposure build the tolerance that sigma-1 agonism was pharmaceutically providing. The existential questions — which belong in the contemplative traditions that have been holding them for millennia and that DXM was providing a shortcut to at the cost of NMDA rebound and hepatotoxicity.

The cough syrup told you the truth: your nervous system needed something. The recovery is finding out what that something actually is — and building the conditions in which your own biology can provide it.

The noise went away when you used DXM. Recovery is learning that the noise was always manageable. The nervous system was built to manage it. You just needed the right tools and someone to take seriously what the cough syrup was actually saying.

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Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE

How to Recover from DXM Using the Recovery Kernel | Substance-Specific Navigation Series